The term campylobacter is derived from Greek words meaning "curved rod." The older literature classified *C jejuni* in the genus *Vibrio* because of morphologic similarities to *V cholera*. Differences in biochemical and growth characteristics have led to the creation of a separate genus, *Campylobacter*. The organisms that cause human enteritis used to be classified as *Campylobacter fetus* subspecies *jejuni*. Recently they have been elevated to a separate species *Campylobacter jejuni*.

Before 1977 *C jejuni* were missed on cultures of stool specimens because of their nonstandard growth requirements. Identification is usually accomplished by using a selective medium containing antibiotics to suppress other intestinal flora in an atmosphere containing 5 percent oxygen and at a temperature of 42°C. Many laboratories will not routinely plant stool cultures under these conditions. Thus a special request to culture for *C jejuni* may have to be made by the physician ordering the culture.

The route of transmission of *C jejuni* is not entirely clear. Fecal-oral transmission and contaminated food and water are considered to be major ways. The organism is widespread among animals, both as commensal gastrointestinal flora and as a cause of enteritis. A large outbreak in Vermont was traced to a contaminated water supply. Dogs with enteritis have been implicated in other studies. Person-to-person transmission, especially among children, is well documented.

Patients of all ages are affected, with peak incidence for children occurring during the first 5 years of life and for adults at 20 to 25 years. Most cases occur during the summer months.

Clinical presentation varies from mild to severe disease. Incubation is usually one to six days. A febrile prodrome with headache, malaise and variable temperature elevation is common. This is followed by abdominal pain and diarrhea that begins with watery and profuse stools. Dysenteric stools develop after a few days. Gross blood and mucus are present in the stools of most patients at some time during the course of the illness. Severe colicky abdominal pain is a hallmark of *Campylobacter* enteritis. The pain can be severe enough to mimic an acute condition of the abdomen. Laparotomies in search of a ruptured viscus are common. Dehydration and vomiting may occur in a few patients. Fecal leukocytes are present in most cases where examinations have been carried out. Diagnosis is made by culture of stool specimens.

Duration of symptoms is usually seven to ten days. Some patients, however, have a relapsing course for several months. *Campylobacter* persists in the stools of patients after recovery but for a shorter period than *Salmonella*. Children in a Montreal study excreted bacteria for an average of 26 days, while 90 percent of Swedish patients had negative cultures after five weeks. Isolation of *C jejuni* from the stool of asymptomatic persons is rare.

C jejuni in vitro are sensitive to several antibiotics, notably erythromycin and tetracycline. Controlled trials have shown that stool cultures become negative and symptoms cease in 48 hours when erythromycin is administered orally. Duration of symptoms was not significantly different in treated and untreated patients when therapy was begun four or more days after the onset of symptoms. However, fecal shedding was considerably shortened by erythromycin treatment. At present antibiotics are usually reserved for patients with severe or protracted illness but may be useful in reducing horizontal transmission.

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Treatment and Prognosis of Reye's Syndrome

REYE'S SYNDROME is an acute noninflammatory encephalopathy associated with fatty change in the liver. It occurs in previously healthy, vigorous children and ranks behind only acute infectious encephalitis as the most common cause of death in virus-related diseases of the central nervous system (CNS). The early recognition of this syndrome by primary care physicians, coupled with organized protocols of therapy, has resulted in decreased mortality in recent years.

Reye's syndrome is heralded by protracted vomiting followed by alternating periods of lethargy and excitability in a child with a trivial, antecedent viral illness. Geographic and temporal clusters are associated with influenzae A and B. Noncluster cases occur throughout the year asso-

ciated with other illnesses such as varicella gastroenteritis. Salicylates have been implicated by some investigators in the pathogenesis of Reye's syndrome, but direct proof is lacking. The diagnosis should be strongly suspected in a nonjaundiced child with a clinical history compatible with Reye's syndrome and SGOT, SGPT and serum ammonia levels greater than twice the upper limits of normal. Supporting laboratory data include a prolonged prothrombin time, serum bilirubin level less than 3 mg per dl, hypoglycemia and normal cerebrospinal fluid. If not clear, the diagnosis may be confirmed by percutaneous liver biopsy that shows microvesicular fat accumulation. Mimicking conditions, including varicella-associated CNS and hepatic dysfunction, can thus be excluded.

Rapid deterioration through progressive stages of coma culminating in irreversible or lethal neurologic damage is usually associated with elevation of intracranial pressures (ICP). Therapy therefore hinges on relief of increased ICP, assessed by an ICP monitoring device, and is most efficiently accomplished in a regional intensive care unit with a critical care team following an anticipatory protocol. Principal modalities for the relief of elevated ICP currently include osmotherapy with mannitol and controlled hyperventilation. Some centers also use hypothermia, short-acting barbiturates and decompressive craniectomy.

These approaches have been associated with a decrease in mortality during the past 5 years from approximately 40 percent to 20 percent or less. The stage of coma at which intensive supportive care is begun appears to influence the prognosis. While progression to deeper stages of coma is common following institution of therapy, admission in stages of deep coma is associated with higher mortality. The incidence of neurologic dysfunction following recovery from Reye's syndrome is not well established. School performance may be affected in children who otherwise show no disability, and severe motor disorders may persist in those who progressed to stages of deep coma.

These considerations prompt the following recommendations: Recognition of Reye's syndrome requires a child's immediate admission to hospital, intravenous administration of 10 percent dextrose and half-normal saline solution at a maintenance rate (1,500 ml per sq meter per 24 hours) and careful monitoring of mental and cardiopulmonary status. Early consultation with and transfer to a referral center familiar with management of Reye's syndrome is appropriate because of the complexity of treatment and implications for a good outcome.

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Decongestants and Antihistamines in the Care of Acute or Serous Otitis Media

DECONGESTANTS AND ANTIHISTAMINES are commonly used in the treatment of otitis. Pneumatic otoscopy and tympanometry have improved a clinician's ability to diagnose serous otitis media (SOM). SOM develops in about 20 percent to 50 percent of children after an episode of acute otitis media (AOM). A child under age two is more likely to have a persistent effusion, but no studies currently indicate the long-term prognosis.

Recently several studies have examined prevention of SOM following AOM by giving drug or placebo along with antibiotic treatment for AOM. Not every study used the objective criterion of tympanometry. When compared with placebo, there was no substantial reduction in the occurrence of SOM after two weeks for pseudoephedrine hydrochloride or sulfate, brompheniramine maleate, tripolidine hydrochloride or a tripolidine-pseudoephedrine (Actifed) combination.

Randall in a similar manner tried to prevent AOM by initiating antihistamine-decongestant (brompheniramine-phenylephrine-phenylpropanolamine [Dimetapp]) treatment when symptoms of an upper respiratory tract infection were felt. When compared with placebo, there was no significant decrease in the development of AOM.

The medical treatment of SOM, once it is established, has been equally frustrating. In their studies, Olson and co-workers kept children with SOM after AOM on a drug or placebo for an additional four to six weeks. Neither pseudoephedrine nor brompheniramine improved the rate of cure. The subgroup who had a past history of SOM actually did worse on either treatment compared with placebo. Fraser and colleagues also failed to show either Dimetapp, ephedrine nose drops or auto inflation (by obstructing the nose and blowing to open the eustachian tube) as useful in British children with SOM. However, children with long-standing SOM may have underlying allergies